Acta Psychiatrica Scandinavica

Acta Psychiatr Scand 2017: 135: 489–498

DOI: 10.1111/acps.12729

Variation in the recall of socially rewarding information and depressive symptom severity: a prospective cohort study

Lewis G, Kounali D-Z, Button KS, Duffy L, Wiles NJ, Munafò MR, Harmer CJ, Lewis G. Variation in the recall of socially rewarding information and depressive symptom severity: a prospective cohort study.

Objective: To test the association between recall for socially rewarding (positive) and/or socially critical (negative) information and depressive symptoms.
Method: Cohort study of people who had visited UK primary care in the past year reporting depressive symptoms (N = 558, 69% female). Positive and negative recall was assessed at three time-points, 2 weeks apart, using a computerised task. Depressive symptoms were assessed at four time-points using the Beck Depression Inventory (BDI). Analyses were conducted using multilevel models.
Results: Concurrently we found evidence that, for every increase in two positive words recalled, depressive symptoms reduced by 0.6 (95% CI −1.0 to −0.2) BDI points. This association was not affected by adjustment for confounders. There was no evidence of an association between negative recall and depressive symptoms (−0.1, 95% CI −0.5 to 0.3). Longitudinally, we found more evidence that positive recall was associated with reduced depressive symptoms than vice versa.
Conclusion: People with more severe depressive symptoms recall less positive information, even if their recall of negative information is unaltered. Clinicians could put more emphasis on encouraging patients to recall positive, socially rewarding information, rather than trying to change negative interpretations of events that have already occurred.

G. Lewis[1], D.-Z. Kounali[2], K. S. Button[3], L. Duffy[1], N. J. Wiles[4], M. R. Munafò[5], C. J. Harmer[6], G. Lewis[1]

[1]Division of Psychiatry, Faculty of Brain Sciences, University College London, London, [2]School of Social and Community Medicine, University of Bristol, Bristol, [3]Department of Psychology, University of Bath, Bath, [4]Centre for Academic Mental Health, School of Social and Community Medicine, University of Bristol, Bristol, [5]MRC Integrative Epidemiology Unit and School of Experimental Psychology, University of Bristol, Bristol, and [6]Department of Psychiatry, University of Oxford, Oxford, UK

Key words: depression; emotional memory; epidemiology

Gemma Lewis, UCL Division of Psychiatry, 6th Floor, Maple House, 149 Tottenham Court Road, London W1T 7NF, UK.
E-mail: gemma.lewis@ucl.ac.uk

Previous presentation
Oral presentation at the European Psychiatric Association, Gothenburg, 2nd December 2016.

Additional information
The original data set for the cohort study is available from Dr Gemma Lewis, University College London.

Accepted for publication March 8, 2017

Significant outcomes

- We found evidence that, as recall for positive socially rewarding information increased, severity of depressive symptoms decreased (a reduction of 0.6 (95% CI −1.0 to −0.2) BDI points for every increase in two positive words recalled).
- In contrast, there was no evidence for an association between recall of negative information and depressive symptoms (−0.1 (95% CI −0.5 to 0.3)).
- Our evidence suggests that people with depressive symptoms appear negative because they forget more positive information, even if their recall of negative information is unaltered. This has implications for our understanding of depression, and for the delivery of psychological therapies which could focus more on encouraging recall of positive socially rewarding information.

Limitations

- Although we have conducted the largest study of emotional processing and depressive symptoms to date, we had a low response rate which might have introduced a selection bias.
- There may have been some practice effects for the incidental recall task, although we saw no clear pattern to suggest that this was occurring.

Introduction

Beck introduced the idea that thinking or cognition was abnormal in depression and that negative self-evaluations, expectancies and memory played a key role in depressive illness (1). Since that time, many studies of people with depression have supported his hypotheses (2).

Studies of cognition and depression have tended to use self-report questionnaires that ask participants to self-rate how they might habitually respond to interpersonal or other events (3). This approach has several limitations. It relies upon a conscious judgement so is susceptible to a mood-congruent response bias (4). Depressive symptoms may therefore decrease the reliability of self-reported cognitive processes and experiences (4). It also restricts investigation to conscious thoughts and behaviours.

More recently there has been investigation of tasks that assess attention, perception and memory more directly. These tasks measure automatic or implicit cognitive processes, which may influence thoughts and behaviours without conscious awareness (5, 6). Investigating these more basic cognitive processes could provide insights into brain mechanisms that might underlie depressive symptoms and the 'higher-level' cognitive phenomena that are apparent in clinical practice.

Studies assessing automatic cognitive processes have found that differences between people with and without depression often centre on emotional or affective information that has a 'value', either positive or negative, and is therefore concerned with reward and punishment. Findings are inconsistent, but there is some evidence that people with depression are more sensitive to punishments (including social criticism) and less sensitive to rewards (including social appraisal) (7). For example, studies report faster more accurate recall of negative than positive information in people with depression compared with healthy controls (8–11). This has supported Beck's idea of a 'negative information processing bias'.

However, a few studies have suggested that depression might be characterised by reduced positive processing rather than increased negative processing (12–14). This supports the idea of an 'optimism bias' (15), that mentally healthy people are unrealistically positive and this 'positive bias' reduces as depressive symptoms increase. So, although people with depression appear more negative, it is unclear whether this is because they are more likely to attend to or remember negative information or less likely to attend to or remember positive information, or whether both are occurring. A better understanding of the cognitive processes underlying depression would improve our understanding of the condition, and influence the delivery of psychological therapies such as cognitive behavioural therapy (CBT).

The majority of previous studies have been case–control designs using small student or clinical samples. The small samples used in previous studies could alone explain inconsistent findings in this area. However, case–control designs are also more prone to selection bias than cohort designs, unless controls are recruited from the same population as cases. If they are not, the distribution of the exposure in cases relative to controls is likely to differ from the distribution in the target population, which would produce a biased effect estimate (16, 17). We are only aware of one

case–control study that was designed to minimise this type of selection bias (18). Case–control studies also preclude an examination of how automatic cognitive processing varies according to the severity of depressive symptoms. An alternative strategy would be to recruit from a single population who have been depressed but who now vary in their depressive symptoms, and study the full range of depressive symptoms. Depression is best viewed along a continuum so this design would allow the severity of depressive symptoms to be investigated in relation to automatic cognitive processing (19).

Another limitation of existing evidence is that, to our knowledge, there have been few longitudinal studies. Existing longitudinal studies have used small samples often recruited from student populations, and results are inconsistent (12, 20–22). Whether alterations in positive/negative processing precede or follow depressive symptoms is therefore unclear. Antidepressants result in improved memory for positive words within a few hours of taking them, and before any clinical response (23–25). This is circumstantial evidence that information processing changes might precede changes in mood.

In this study, we used a large ($N = 558$) prospective longitudinal cohort of people who had presented to UK primary care surgeries with depression in the previous year. To broaden inclusion criteria, participants were selected if they had reported depressive symptoms, disorder or depressed mood. We tested concurrent and longitudinal associations between depressive symptoms and memory for socially rewarding (positive) and socially critical (negative) information, as a measure of automatic emotional processing.

Method

Participants

The sample was recruited from primary care surgeries in three UK sites (Bristol, Liverpool, and York). The primary aim of the study was to examine the relationship between self-reported improvement and scores on standardised assessments of depression. This study is a secondary analysis. Computerised records were searched at each site to identify people who had reported depressive episodes, depressed mood, depressive symptoms or a major depressive episode in the past year. Individuals were included if they were aged between 18 and 70 years, treated or not treated with antidepressants and referred or not referred to improving access to psychological therapies (IAPT) services. We excluded people who were diagnosed with bipolar disorder, psychosis or an eating disorder; had alcohol or substance use problems; were unable to complete study questionnaires; or were 30 weeks or more pregnant. The remainder ($N = 7721$) were sent an information letter in the post, and 1470 (19%) replied. Of these, 821 (55%) were willing to be contacted, 23 (3%) of whom were ineligible. The remaining 798 were contacted to arrange an interview, and 563 (71%) consented. Interviews were conducted at four time-points, two weeks apart, at the participants' home or GP surgery. At time one, 558 people provided data (five could not be contacted), and at follow-ups two, three, and four: 476 (85%); 443 (79%) and 430 (77%). All participants provided written informed consent, and ethical approval was obtained from NRES Committee South West-Central Bristol. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

Measures

Depressive symptoms. The Beck Depression Inventory (BDI-II) (26) is a 21-item self-report measure of the severity of depressive symptoms and was completed at times one, two, three and four. Scores ranged from 0 to 58, with higher scores indicating greater severity. Internal consistency was high at each time-point (Cronbach's alpha ranging from 0.93 to 0.95). The Clinical Interview Schedule Revised (CIS-R) (27) was administered at baseline (only) and can be used to derive diagnoses of current depression based on symptoms occurring in the past week, according to the International Classification of Diseases (ICD-10).

Memory for socially rewarding and socially critical information. This was assessed at times one, two and three, using a computerised task administered by researchers at the participants' home or primary care surgery. At each time-point, twenty likeable (e.g. cheerful, honest and optimistic) and twenty dislikeable (e.g. domineering, untidy and hostile) personality characteristics were presented on a computer screen in a random order (each word was presented for 500 milliseconds) (23, 28). A different set of words was presented at each time-point, and words were randomised within time-points (120 words in total). Words were matched

according to length, ratings of usage frequency, and meaningfulness. After each word, participants indicated whether they would 'like' or 'dislike' to overhear someone describing them in this way (by pressing a key on the keyboard), so the task was socially and self-relevant. At the end of the task, participants were asked to recall as many words as possible in 2 min. This was a surprise recall task (at the first time-point), to test incidental memory. The number of positive and negative words accurately recalled (hits), and the number of false responses (intrusions) were computed.

Confounders. Demographic variables that were associated with depression and recall ability were ascertained by self-administered computerised questionnaire at baseline (the CIS-R). The CIS-R was administered by researchers at the participants' home or primary care surgery. Confounders were age, sex, education level and negative life events, based on evidence that they were associated with both recall and depressive symptoms and could therefore be common causes. Negative life events were assessed using eight items which included bereavement, separation or divorce and illness or injury. A binary variable was created (0 = one or none; 1 = more than one). Education was rated using seven categories ranging from 'no qualifications' to 'higher degree', higher values indicating higher education levels. A binary variable was created (0 = lower education, 1 = higher education). We also adjusted for antidepressant use at times 1–3 using a binary variable (0 = not taking antidepressants, 1 = taking antidepressants).

Statistical analyses

Concurrent associations. Linear multilevel mixed-effects regression models (LMMs) were used to analyse concurrent associations between positive and negative hits (continuous exposures) and depressive symptoms (continuous outcome). Analysis of repeated measures data using LMMs allows the use of all available data and increases the precision of estimates. Positive and negative hits were included in the same model to adjust for recall ability. Models are presented before and after adjustment for confounders. We also tested a Poisson multilevel mixed-effects model using number of words (positive and negative) correctly recalled as the outcome variable, and depressive symptoms and valence (positive or negative) as exposure variables. This allowed us to test for an interaction between depressive symptoms and valence, to establish whether the association between positive recall and depressive symptoms was stronger than the association between negative recall and depressive symptoms. This model was run before and after adjustment for confounders.

Longitudinal associations. We computed longitudinal trajectories for positive and negative recall and depressive symptoms, to create a multilevel joint model across period-lagged occasions (29). Advantages of this approach include use of data from all time-points and acknowledgement of the correlated nature of repeated measures (30). It is particularly useful when there are missing values and measurement error. The recall variable was the proportion of positive hits (of the total number of positive and negative hits), modelled on a standardised scale; one standard deviation corresponded to 14% positive words recalled. Similarly, depressive symptoms were modelled on a standardised scale, one standard deviation corresponded to 14 BDI points. Each trajectory included a linear random intercept and slope (random effects) that captured between individual variation around average baseline levels and change (31, 32). Random intercepts are interpreted as individual latent baseline levels. Random slopes are interpreted as individual change over time (Figure S2).

Estimation of the variance-covariance matrix at the individual level allowed us to perform post-estimation calculations. These quantified conditional expectations for depressive symptoms as a function of individuals' previous recall and changes in recall and vice versa (recall as a function of depressive symptoms, to test the reverse hypothesis that depressive symptoms precede changes in recall). Estimation was carried out by Markov Chain Monte Carlo (MCMC) using WinBUGs (33). Convergence, based on statistical criteria, was achieved within 50 000 iterations (34). Posterior summaries were based on 100 000 samples over five chains with different starting values, and after discarding the first 200 000 samples. This allowed us to arrive at estimates with Monte Carlo error less than 5% of the sample standard deviation for all parameters (34). Stochastic simulation through MCMC allowed us to compute Bayesian full posterior distributions of effects as opposed to summaries, for example, means and standard errors under assumptions of normality derived from asymptotic arguments. We first report the 'most likely value' for the strength of associations, which is akin to a point estimate in frequentist approaches. We also report the 95% credible interval (CrI) for the most likely value, which is akin to a confidence

Table 1. Sample characteristics and mean (SD) number of positive and negative words correctly recalled, $N = 534$

Characteristic	Positive words	*P* value	Negative words	*P* value
Age				
Under 50 (276)	2.8 (1.9)		2.0 (1.5)	
Over 50 (258)	1.9 (1.6)	<0.0001	1.3 (1.2)	<0.0001
Gender				
Male (168)	2.0 (1.6)		1.3 (1.2)	
Female (366)	2.6 (1.9)	0.0014	1.8 (1.4)	0.0002
Education				
Lower (205)	1.9 (1.6)		1.2 (1.2)	
Higher (329)	2.7 (1.8)	<0.0001	1.9 (1.5)	<0.0001
Currently taking antidepressants				
Yes (370)	2.4 (1.9)		1.6 (1.4)	
No (164)	2.3 (1.8)	0.9927	1.7 (1.4)	0.5423
Life events				
None (225)	2.2 (1.7)		1.7 (1.4)	
One or more (309)	2.5 (1.8)	0.0908	1.6 (1.4)	0.7958
Long-standing physical illness				
Yes (385)	2.6 (1.9)		1.8 (1.5)	
No (149)	2.3 (1.7)	0.1077	1.6 (1.3)	0.1516
Depression diagnosis				
Yes (241)	2.2 (1.8)		1.6 (1.4)	
No (293)	2.6 (1.8)	0.0241	1.7 (1.4)	0.4776

Table 2. Descriptive statistics of modelled variables: depressive symptoms (BDI-II), positive and negative words correctly recalled (hits) and proportion of positive words correctly recalled

	Time-point	Mean	SD
Depressive symptoms	1	20.1	12.2
	2	17.7	12.4
	3	15.9	12.6
	4	15.6	12.8
Positive hits	1	2.4	1.8
	2	2.7	2.0
	3	2.0	1.6
Negative hits	1	1.7	1.4
	2	1.6	1.4
	3	1.6	1.6
Proportion of hits positive	1	0.6	0.1
	2	0.6	0.1
	3	0.5	0.2

interval. We then report a range of centile values for these distributions, along with probabilities quantifying the likelihood of 'no effect or an association in the opposite direction' (Bayesian *P*-values).

Results

Descriptive statistics

We excluded participants ($N = 24$) who were missing baseline demographics, or data on recall or depressive symptoms at all time-points. Participants with missing data had higher baseline depressive symptoms. They did not differ on the exposure variable, recall. The final sample comprised 534 participants (96% of total sample) aged between 18 and 76 years (mean 48.4). At baseline, depressive symptoms ranged from 0 to 58 (Figure S1). Mean positive and negative words recalled are shown in Table 1, according to demographic and clinical characteristics. Mean depressive symptoms and recall scores over time are shown in Table 2. Overall, people remembered more positive than negative words (mean difference 0.72, 95% CI 0.60–0.84, $P < 0.001$). The results were unaltered when analyses were conducted on a sample with complete data on exposures and outcome at all time-points ($N = 393$, available on request).

Table 3. Reduction in BDI scores (outcome) for every 1 standard deviation (2-point) increase in positive and negative words correctly recalled (concurrent associations)

	Model 1: unadjusted ($N = 530$)			Model 2: adjusted for confounders* ($N = 524$)		
	Coef	95% CI	*P*	Coef	95% CI	*P*
Positive hits	−0.58	−1.0 to −0.15	0.009	−0.61	−1.1 to −0.17	0.006
Negative hits	−0.10	−0.51 to 0.31	0.629	−0.11	−0.52 to 0.30	0.604

*Confounders were age, sex, education, antidepressant use and negative life events.

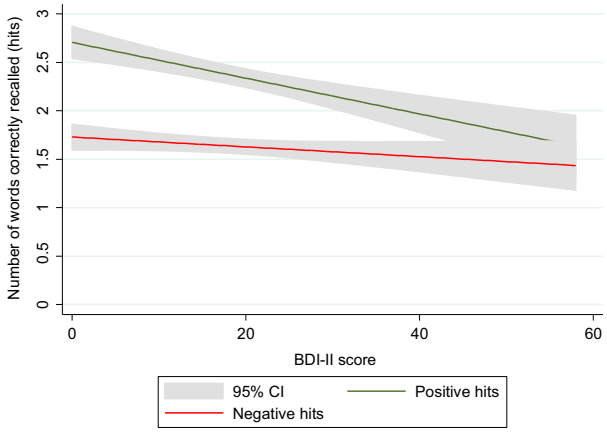

Fig. 1. Association between depressive symptoms (BDI-II score) and number of positive (green line) and negative (red line) words correctly recalled (hits). $N = 524$.

Concurrent associations

Concurrent associations between recall and depressive symptoms are displayed in Table 3. For every 2-point increase in the number of positive words recalled, BDI scores reduced by around half a point (95% CI −1.0 to −0.15). There was no evidence for an association between recall of negative words and depressive symptoms, except before

adjustment for positive recall (Fig. 1). The association between positive recall and depressive symptoms was not affected by adjustment for confounders (Table 3). There was evidence of an interaction between depressive symptoms and valence, so the inverse association between recall and depressive symptoms was stronger for positive than negative words (interaction term after adjustment for confounders: coefficient 0.005, 95% CI 0.0004 to 0.01, $P = 0.032$).

Antidepressant use

Antidepressant use was stable over time (correlation coefficients ranged from 0.94 to 0.97). Participants taking antidepressants had more severe depressive symptoms (mean difference 4.1, 95% CI −5.6 to −2.7, $P < 0.0001$) and recalled slightly more positive words (Table 1), but this difference was small and weak (coefficient −0.00, 95% CI −0.34 to 0.34, $P = 0.993$). There was no evidence that associations between recall and depressive symptoms differed according to antidepressant use (interaction term for positive recall and antidepressant use: coefficient −0.31, 95% CI −0.84 to 0.21, $P = 0.237$; negative words: coefficient 0.56, 95% CI −0.07 to 1.18, $P = 0.081$). At time one and time two, adjustment for antidepressant use resulted in a slight increase in the strength of the inverse association between positive recall and depressive symptoms (Table S1).

Longitudinal associations

The most likely values, credible intervals and probability distributions (2.50–97.50%) are displayed in Table 4. Models 1 and 2 show expected values for subsequent depressive symptoms, as a function of the previous proportion of positive words recalled (before and after adjustment for confounders). A one standard deviation increase in baseline proportion of positive words recalled (model 1: baseline EP levels) was associated with a reduction in depressive symptoms of 1.09 standard deviations or 15 BDI points (95% CrI −3.04 to 0.85). There was a small probability (only 8%) that this estimated value was 0 (evidence of no association) or greater (evidence that depressive symptoms increased after recall of positive words increased). After adjustment for confounders, the most likely value was slightly attenuated at −0.93 (13 BDI points), the associated errors slightly larger and the resulting credible intervals wider (Table 4: model 2, Baseline EP levels). For individuals whose recall scores were average at baseline but increased over time, there was weaker evidence of a reduction in depressive symptoms (Table 4: model 1, EP increase). For a one standard deviation increase in the proportion of positive words recalled from baseline, the average expected BDI score decreased by 0.83 of a standard deviation (12 BDI points). However, uncertainty associated with this estimate was greater (95% CrI −3.59 to 1.82) (Table 4: model 1, EP increase). These results are depicted in Figure S3.

In Table 4, models 3 and 4 show results from the reverse tests, that depressive symptoms occurred before any change in recall. There was no evidence that depressive symptoms were associated with subsequent proportion of positive words recalled. These results are depicted in Figure S4, which shows that the probability that the association between previous depressive symptoms and subsequent positive recall was equal to or more than zero was 20%.

In summary, when both processes (recall and depressive symptoms) were modelled simultaneously, we found more evidence that positive recall

Table 4. Longitudinal associations between proportion of positive words correctly recalled (emotional processing, EP) and depressive symptoms (BDI-II scores), $N = 534$

				Probability level (%)					
Model	Parameters	Most likely value	SD	2.50	5.00	10.00	90.00	95.00	97.50
Model 1. Conditional expectation of EP as a function of BDI									
	Baseline EP	−1.09	0.90	−3.04	−2.69	−2.25	−0.22	0.13	0.85
	EP increase	−0.83	1.34	−3.59	−3.08	−2.50	0.87	1.37	1.82
Model 2. Model 1 adjusted for confounders*									
	Baseline EP	−0.93	0.73	−2.68	−2.24	−1.85	−0.14	0.16	0.41
	EP increase	−0.49	1.22	−3.16	−2.70	−2.11	0.93	1.43	1.79
Model 3. Conditional expectation of BDI as a function of EP									
	Baseline BDI	−0.12	0.15	−0.43	−0.36	−0.11	0.05	0.12	0.18
	BDI increase	0.31	1.54	−2.83	−2.24	0.28	2.26	2.91	3.46
Model 4. Model 3 adjusted for confounders*									
	Baseline BDI	−0.11	0.15	−0.42	−0.36	−0.29	0.06	0.13	0.19
	BDI increase	0.25	1.58	−3.08	−2.45	−1.73	2.23	2.87	3.41

*Confounders were age, sex, education and antidepressant use.

was associated with reductions in subsequent depressive symptoms than the other way around. This was still the case after adjusting for confounders, although there was some attenuation.

Discussion

We found evidence that, as recall of socially rewarding words increased, severity of depressive symptoms decreased, but recall of socially critical words was relatively unaltered. This suggests that depression is characterised by reduced positive or reward processing rather than increased negative or punishment processing. The apparent negativity of depression might therefore result from less positivity rather than increased negativity. In longitudinal analyses we found some evidence, albeit weak, that reduced recall of socially rewarding words preceded depressive symptoms. There was no evidence in the opposite direction, that depressive symptoms or how they changed preceded recall. The longitudinal associations we observed were large, but there was 8% probability that the association was actually zero or going in the opposite direction. However, it is notable that these temporal associations were found in an observational study where changes in depressive symptoms and memory were modest.

Strengths and limitations

To our knowledge, this is the largest study so far to assess automatic emotional processing and depressive symptoms. Our sample included the full range of depressive symptom severity (from no symptoms to many), more likely to reflect that in the community. Unlike previous studies, our sample therefore contained people with no to few symptoms and people with very severe symptoms (e.g. BDI-II score >30). A strength of our approach is that participants with no/few depressive symptoms were recruited from the same population as participants with many symptoms. We therefore think that our comparison between lower and higher severity of depression will lead to more robust conclusions than a case–control design in which those without depression are selected in a different way to those with depression. Our low response rate is a limitation, but we think that the factors influencing selection bias will be similar for the higher and lower scorers, although we of course cannot be certain of this. We also adjusted for several potential confounders which did not alter our findings, and our sample was naturalistic in that people may or may not have been on antidepressants or receiving psychological therapy.

Another strength of our study was the prospective longitudinal design with repeated measures. Repeated measures increase the precision of estimates which, in a cross-sectional study, would be based on a single snapshot in time. Our estimates also took account of any changes in depressive symptoms and recall between the follow-up assessments. Finally, our statistical methods minimised the influence of missing data (we only excluded participants who were missing data at all time-points), acknowledged correlations between repeated measures and compared reciprocal associations.

Our study has several limitations. First, although we recruited a large sample, we had a low response rate. This would affect representativeness and might have introduced a selection bias. However, as our selection of participants did not depend on the exposure, we think it would be unlikely to have biased the association we observed. Representativeness is also less important when studying mechanisms that can be assumed to apply beyond the study sample (35).

Practice effects are another potential limitation. Even though different words were used at each assessment, after the first assessment, people would have expected the incidental recall task. This may have resulted in increased recall. However, there was no consistent pattern to suggest this had occurred.

We also cannot be sure that our results will extend to other cognitive tasks concerned with reward and punishment. Similarly, we cannot identify whether reduced positive biases are associated with the encoding, storage or retrieval of emotional information. Finally, as with all observational studies, we cannot provide evidence of a causal effect of reduced positive processing on depressive symptoms. Although we adjusted for several potential confounders, which did not alter our association, residual confounding is still a possibility.

Would receipt of treatment confound the observed relationship?

There was no evidence that antidepressants confounded the association. Antidepressants in within-subject experimental studies lead to an increase in positive recall (23). We found some evidence for this in our study, although it was very weak. Antidepressant use was commoner in those with more depressive symptoms so if this were to confound the relationship, it would have strengthened the negative association rather than resulting in a spurious one (36). In fact, we did find a slight strengthening of the association when we adjusted for antidepressants.

Information on psychological therapies was unavailable in our sample. The cognitive neuropsychological model proposes that psychological therapies, like antidepressants, increase positive processing (5, 37). This is supported by two small studies ($N < 58$) using mindfulness training and mindfulness-based cognitive therapy that reported increased recall for positive information (38, 39). We would expect that, if psychological therapies were to confound this relationship, the effect would be similar to that of antidepressants. Adjustment for psychological therapies would therefore be expected, if anything, to increase the negative association between positive recall and depressive symptoms (36).

Our results suggest that the negative information processing observed in people with depression may result more from a reduction in positive than an increase in negative input. If there is less positive information available for retrieval, or a similar amount of positive and negative information (i.e. no positive 'bias'), negative information might be more easily retrieved. This could explain why people with depression often appear to recall negative information (4). Our longitudinal findings suggest that reduced positive recall does not result from depressed people withdrawing from positive experiences and positive information becoming less salient. Instead, our findings suggest that reduced positive processing may precede depression. This is consistent with a smaller longitudinal study, which found that increased positive recall was associated with later reductions in depressive symptoms, with no evidence of an association with negative recall (12).

Recall of positive over negative information may enhance mental health by providing more positive predictions of future events. For example, if people are more likely to recall positive than negative social interactions, they are more likely to engage in future social interactions (15). This could result in higher levels of social activity and increased social reward. Short-term recall of socially rewarding information might also lead to the development of more positive longer-term memories about the self. This would increase the amount of socially rewarding information that is available for future recall. Our findings point to the importance of recall of specific rather than overgeneral positive information. This could reduce overgeneral autobiographical memory, which has been identified as a risk factor for depression (40, 41). Recall of more positive information might also lead to more positive thoughts and emotions, which have been associated with well-being (42). Increased positive processing may also reduce rumination, which has been associated with depressive symptoms (43, 44).

Our sample included people who were taking antidepressants and antidepressants have been found to affect positive recall (23). This effect is seen with serotonin reuptake inhibitors, reboxetine and mirtazapine. However, even though we found that people on antidepressants recalled more positive words, this association was small and weak. The evidence that antidepressants affect recall of positive words arises from experimental designs that studied within subject differences. These designs would provide more precise estimates and reduce confounders. We know of another observational study similar to ours and they also report no evidence for an association between antidepressant use and positive recall (12).

Our findings are relevant to the observation that antidepressants improve recall of positive socially rewarding words but do not affect recall of negative socially critical words (23). This effect of antidepressants is the reverse of our observation that people with more depressive symptoms had less positive recall without any evidence for a change in negative recall. Together, these results support the hypothesis that increased positive processing may be one of the mechanisms underlying the treatment effects of antidepressants.

If depression is characterised by reduced positive processing rather than increased negative processing, our findings are, at first sight, at odds with Beck's theory of how CBT works. However, in CBT, patients are encouraged to reappraise events, so CBT could also reinstate the positive bias that is a sign of better mental health. The emphasis on the positive rather than the negative is also a feature of other therapies such as mindfulness, behavioural activation, emotion focused therapy, and acceptance and commitment therapy. Our findings are that people with depression recall less positive information, even if their recall of negative information is unaltered. This suggests that even if a person is unable to modify their negative cognitions, increased positive processing might still improve their depressive symptoms. Clinicians, for example primary care practitioners and therapists, could therefore put more emphasis on encouraging patients to recall positive, socially rewarding information, rather than trying to change negative interpretations of events that have already occurred.

Acknowledgements

We would like to thank the participants who took part in the study, and the general practice surgeries for their help with

recruitment. We also thank the research team: researchers, research nurses and administrative staff.

Funding

This article summarises independent research funded by the National Institute for Health Research (NIHR) under its Programme Grants for Applied Research, Reference Number RP-PG-0610-10048. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health. The funding source had no role in study design, data collection, data analysis, interpretation or writing of the report. The corresponding author had full access to all data used in the study and had final responsibility for the decision to submit for publication.

Declaration of interest

None.

References

1. Beck A. Depression: clinical, experimental, and theoretical aspects. New York: Harper & Row; 1967.
2. Beck A. The evolution of the cognitive model of depression and its neurobiological correlates. Am J Psychiatry 2008;**165**:969–977.
3. Elliott R, Zahn R, Deakin JFW, Anderson IM. Affective cognition and its disruption in mood disorders. Neuropsychopharmacology 2011;**36**:153–182.
4. Colman I, Kingsbury M, Garad Y et al. Consistency in adult reporting of adverse childhood experiences. Psychol Med 2016;**46**:543–549.
5. Roiser JP, Elliott R, Sahakian BJ. Cognitive mechanisms of treatment in depression. Neuropsychopharmacology 2011;**37**:117–136.
6. Kahneman D. Thinking fast and slow. New York: Farrar, Straus and Giroux; 2011.
7. Eshel N, Roiser JP. Reward and Punishment Processing in Depression. Biol Psychiatry 2010;**68**:118–124.
8. Bradley BP, Mogg K, Millar N, White J. Selective processing of negative information: effects of clinical anxiety, concurrent depression, and awareness. J Abnorm Psychol 1995;**104**:532–536.
9. Bradley BP, Mogg K, Williams R. Implicit and explicit memory for emotional information in non-clinical subjects. Behav Res Ther 1994;**32**:65–78.
10. Direnfeld DM, Roberts JE. Mood congruent memory in dysphoria: the roles of state affect and cognitive style. Behav Res Ther 2006;**44**:1275–1285.
11. Dunbar GC, Lishman WA. Depression, recognition-memory and hedonic tone a signal detection analysis. Br J Psychiatry 1984;**144**:376–382.
12. Johnson SL, Joormann J, Gotlib IH. Does processing of emotional stimuli predict symptomatic improvement and diagnostic recovery from major depression? Emotion 2007;**7**:201–206.
13. Garrett N, Sharot T, Faulkner P, Korn CW, Roiser JP, Dolan RJ. Losing the rose tinted glasses: neural substrates of unbiased belief updating in depression. Front Hum Neurosci 2014;**8**:639.
14. Gilboa E, Roberts JE, Gotlib IH. The effects of induced and naturally occurring dysphoric mood on biases in self-evaluation and memory. Cogn Emot 1997;**11**:65–82.
15. Sharot T. The optimism bias. Curr Biol 2011;**21**:R941–R945.
16. Lewis G, Pelosi AJ. The case-control study in psychiatry. Br J Psychiatry 1990;**157**:197–207.
17. Rothman K, Greenland S, Lash T. Modern epidemiology. Third. Philadelphia, PA: Lippincott Williams & Wilkins, 2013.
18. Dunbar GC, Lishman WA. Depression, recognition memory and hedonic tone: a signal detection analysis. Br J Psychiatry 1985;**144**:376–382.
19. Hankin BL, Fraley RC, Lahey BB, Waldman ID. Is depression best viewed as a continuum or discrete category? A taxometric analysis of childhood and adolescent depression in a population-based sample. J Abnorm Psychol 2005;**114**:96–110.
20. Rude SS, Valdez CR, Odom S, Ebrahimi A. Negative cognitive biases predict subsequent depression. Cognit Ther Res 2003;**27**:415–429.
21. Everaert J, Duyck W, Koster EHW. Emotionally biased cognitive processes: the weakest link predicts prospective changes in depressive symptom severity. PLoS ONE 2015;**10**:1–9.
22. Tranter R, Bell D, Gutting P, Harmer CJ, Healy D, Anderson IM. The effect of serotonergic and noradrenergic antidepressants on face emotion processing in depressed patients. J Affect Disord 2009;**118**:87–93.
23. Harmer CJ, O'Sullivan U, Favaron E et al. Effect of acute antidepressant administration on negative affective bias in depressed patients. Am J Psychiatry 2009;**166**:1178–1184.
24. Harmer CJ, Bhagwagar Z, Perrett DI, Völlm BA, Cowen PJ, Goodwin GM. Acute SSRI administration affects the processing of social cues in healthy volunteers. Neuropsychopharmacology 2003;**28**:148–152.
25. Harmer CJ, Shelley NC, Cowen PJ, Goodwin GM. Increased positive versus negative affective perception and memory in healthy volunteers following selective serotonin and norepinephrine reuptake inhibition. Am J Psychiatry 2004;**161**:1256–1263.
26. Beck A, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psychiatry 1961;**4**:561–571.
27. Lewis G, Pelosi AJ, Araya R, Dunn G. Measuring psychiatric disorder in the community: a standardized assessment for use by lay interviewers. Psychol Med 2009;**22**:465–486.
28. Anderson NH. Likableness ratings of 555 personality-trait words. J Pers Soc Psychol 1968;**9**:272–279.
29. Guo X, Carlin BP. Separate and joint modeling of longitudinal and event time data using standard computer packages. Am Stat 2004;**58**:16–24.
30. Shahab L, Gilchrist G, Hagger-Johnson G, Shankar A, West E, West R. Reciprocal associations between smoking cessation and depression in older smokers: findings from the English Longitudinal Study of Ageing. Br J Psychiatry 2015;**207**:243–249.
31. Diggle P. Dealing with missing values in longitudinal studies. In: Everitt BS, Dung G, eds. Statistical analysis of medical data: new developments. London: Hodder Education, 1998:203–228.
32. Diggle PJ. An approach to the analysis of repeated measurements. Biometrics 1988;**44**:959–971.
33. Lunn D, Spiegelhalter D, Thomas A, Best N. The BUGS project: evolution, critique and future directions. Stat Med 2009;**28**:3049–3067.

34. Brooks S, Gelman A. Alternative methods for monitoring convergence of iterative simulations. J Comput Graph Stat 1998;**7**:434–455.
35. Rothman KJ, Gallacher JEJ, Hatch EE. Why representativeness should be avoided. Int J Epidemiol 2013;**42**:1012–1014.
36. Mehio-Sibai A, Feinleib M, Sibai TA, Armenian HK. A positive or a negative confounding variable? A simple teaching aid for clinicians and students. Ann Epidemiol 2005;**15**:421–423.
37. Harmer CJ, Goodwin GM, Cowen PJ. Why do antidepressants take so long to work? A cognitive neuropsychological model of antidepressant drug action. Br J Psychiatry 2009;**195**:102–108.
38. Roberts-Wolfe D, Sacchet M, Hastings E, Roth H, Britton W. Mindfulness training alters emotional memory recall compared to active controls: support for an emotional information processing model of mindfulness. Front Hum Neurosci 2012;**6**:1–13.
39. van Vugt MK, Hitchcock P, Shahar B, Britton W. The effects of mindfulness-based cognitive therapy on affective memory recall dynamics in depression: a mechanistic model of rumination. Front Hum Neurosci 2012;**6**:257.
40. Conway MA, Pleydell-Pearce CW, Pleydell-Pearce W. The construction of autobiographical memories in the self-memory system. Psychol Rev Me Adams Mikulincer Woike 2000;**107**:261–288.
41. Sumner JA, Griffith JW, Mineka S. Overgeneral autobiographical memory as a predictor of the course of depression: a meta-analysis. Behav Res Ther 2010;**48**:614–625.
42. Fredrickson BL, Joiner T. Positive emotions trigger upward spirals toward emotional well-being. Psychol Sci 2002;**13**:172–175.
43. Button KS, Kounali D, Stapinski L, Rapee RM, Lewis G, Munafò MR. Fear of negative evaluation biases social evaluation inference: evidence from a probabilistic learning task. PLoS ONE 2015;**10**:e0119456.
44. Wilkinson PO, Croudace TJ, Goodyer IM. Rumination, anxiety, depressive symptoms and subsequent depression in adolescents at risk for psychopathology: a longitudinal cohort study. BMC Psychiatry 2013;**13**:250.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Figure S1. Distribution of BDI-II scores at baseline.

Figure S2. Directed acyclic graph (DAG) for hypothesized longitudinal associations between depressive symptoms (BDI-II scores) and emotional processing (EP).

Figure S3. Conditional expectations for proportion of positive words recalled as a function of depressive symptoms, after adjustment for confounders (age, sex, education, antidepressant use).

Figure S4. Conditional expectations for depressive symptoms as a function of proportion of positive words recalled, after adjustment for confounders (age, sex, education, antidepressant use).

Table S1. Associations between number of positive words correctly recalled (hits)and depressive symptoms (outcome variable), before and after adjustment for antidepressant use.